THE RECOVERY KERNEL

THE IMPLEMENTATION GUIDE

From Whole-Genome Sequencing to Daily Stack — How to Actually Run the Framework

A practitioner-level walkthrough of the decision tree. Your DNA sets the foundation. Your substance sets the counter-move. The two together are the protocol.

⚠ **Read first**

This is a harm-reduction and education resource — not medical advice, diagnosis, or prescription. Nothing here replaces a physician, pharmacist, or prescriber.

Crisis: call or text 988. Medical emergency: call 911. Alcohol, benzodiazepine, and (for GABRB3-risk individuals) GABA-depleting withdrawal can cause fatal seizures — never stop or taper those without medical supervision. Contact a physician before stopping, not after.

Every supplement here can interact with medications. Run every addition through a free interaction checker (Drugs.com, Medscape) and confirm with a pharmacist before starting.

How to use this guide

The rest of the library assumes you already know how to answer one question: once you accept that addiction vulnerability is partly genetic, what do you actually do — day to day — given your specific DNA and your specific substance? This document is that answer, built as a decision tree you move through in order.

- **Step 1 — Get the data.** Whole-genome sequencing → a VCF file → the domain profiler. If you can't sequence yet, the free phenotype self-assessments stand in.
- **Step 2 — Read the six gene systems.** MTHFR (supply), COMT (clearance), CYP2D6 (processing), the GABRA2/GABRB3 braking system (impulse control), and the OPRM1/DRD2 receptor layer. Each has a few possible results, and each result changes your protocol.
- **Step 3 — Resolve your combined profile.** The systems interact multiplicatively. The guide names the recognized high-risk combinations.
- **Step 4 — Layer in the substance.** The same profile recovers differently from a stimulant vs. a dissociative vs. an opioid. Worked examples: cocaine, ketamine, kratom.
- **Step 5 — Build the stack, taper, and life design** — including the honest timeline adjustment for a substance-damaged brain, and the repair tier the standard stack leaves out.
- **Step 6 — Drive it with AI.** Turn all of the above into a profile you paste into an AI assistant so the guidance is personalized, not generic.

> **The one rule that governs everything below**
>
> **Biology first, taper second, behavior third.** Restoring the neurochemical foundation must precede — or at minimum accompany — stopping the substance. Removing the drug without replacing what it was chemically providing is the mechanism of relapse, not a failure of willpower.

Step 1 — Get the data

1a. The ideal input: sequencing → VCF → profiler

The framework is designed around a piece of tooling: a whole-genome sequence produces a VCF file, a profiler reads that file and outputs domain codes, and a dashboard turns those codes into your matched stack. Here's the actual pipeline:

Test	What it gives you	Role in the framework
30× Whole Genome Sequencing (Nebula, Dante, Sequencing.com)	Full coverage; a VCF file — the raw input the profiler needs. ~$200–400, one time.	Recommended primary. The VCF is a one-time asset that never changes — treat it like a financial document.
Consumer SNP array (23andMe, Ancestry)	~700k pre-selected SNPs as a .txt; covers MTHFR, COMT, DRD2, some CYP.	Acceptable starting point. Doesn't produce a VCF, but enough to begin. Upload to Genetic Genie (free) or Promethease (~$12).

Test	What it gives you	Role in the framework
Clinical pharmacogenomic panel (GeneSight, Mayo GeneGuide)	Clinical-grade CYP2D6/2C19/3A4 calls, physician-ordered.	Valuable add-on for the medication/interaction step; doesn't replace WGS for the full profile.
Blood methylation markers (homocysteine, B12, RBC folate)	Functional proxies, orderable without a physician in most US states.	Quarterly monitoring. Homocysteine is the single most actionable marker.

The profiler + dashboard: the VCF runs through the domain profiler, which scores ~16 biological domains (neuromodulatory, mood-anxiety, detox-liver, cardiovascular, energy-mitochondrial, and so on). Those codes import into the dashboard, which outputs your weight-dosed supplement stack. The principle the library states plainly: implement that output — and only that output — rather than generic assumptions.

The honest leverage move: if you're about to be prescribed anything psychoactive, ask the prescriber to order pharmacogenomic testing *before* they write the script. CYP2D6 status is FDA-labeled as relevant to 60+ medications — a legitimate billable question, not a favor you're conning them into.

1b. If you can't test yet: the free phenotype estimates

You are not blocked. Each gene system below has a validated self-assessment (Book 1's Chapter 2/3 checklists, the Compendium's Protocol 06, and the braking-system assessment in the GABA chapter). They use observable patterns — family history, drug reactions, impulse and anxiety markers — to get you a working hypothesis in ~15 minutes, free. They get you roughly 60–80% of the way to the genetic answer; treat the result as provisional and upgrade it when you sequence. It is a starting point, not a final call — but enough to begin safely.

Step 2 — Read the six gene systems

Single genes are the vocabulary; the combination is the sentence. Here is each system, every result it can return, and what each result changes.

System 1 — MTHFR (the supply line)

Sets how fast you convert folate into the active form your brain uses to build neurotransmitters. A slow variant bottlenecks the whole production line. MTHFR is always treated first — it addresses more vulnerability than any single other intervention, and ~40% of people carry a meaningful variant.

Result	Meaning	What it changes
C/C (wild type)	~100% activity.	No methylfolate protocol typically needed; general hygiene only.
C/T (heterozygous)	~60–65% activity.	Benefits from methylfolate support, especially under stress.
T/T (homozygous)	~30–40% activity; significant bottleneck.	Methylfolate protocol strongly indicated — a foundation, not an add-on.
Compound het (C/T + A1298C A/C)	Effects multiply; behaves like T/T.	Treat as T/T for supplementation.

The non-negotiable MTHFR rule

Any T variant → avoid synthetic folic acid (in fortified foods and cheap multivitamins). A compromised enzyme can't convert it; it accumulates and blocks the sites that would process real folate, quietly worsening the bottleneck. Read every label. Use methylfolate / L-methylfolate / 5-MTHF / Quatrefolic.

System 2 — COMT (the clearance system: Warrior vs Sage/Worrier)

Sets how fast you clear dopamine and stress chemistry from the prefrontal cortex — the single most useful gene for predicting which direction someone's use runs. (The library uses both 'Worrier' and 'Sage' for the slow-clearance Met/Met type; same genotype.)

Result	Profile	Substance lean & recovery need
Val/Val	Warrior — fast clearance	Low baseline dopamine; ordinary life feels flat. Leans stimulant. Needs stimulation, challenge, stakes — not more calm.
Met/Met	Sage / Worrier — slow clearance	Stress chemistry accumulates and floods. Leans depressant. Needs calm, structure, low stimulation — not more intensity.
Val/Met	Balanced	Adaptable; direction set by stress load and other variants.

This is the fork the whole life-design half of the framework hangs on: **the exercise, work environment, and social structure that heal a Warrior actively harm a Sage, and vice versa.**

System 3 — CYP2D6 (the processing system)

The liver enzyme that processes most opioids and many antidepressants. Your status decides whether a standard dose is useless, correct, or dangerous — and explains many past 'drug-seeking' labels that were actually therapeutic failures.

Result	What happens	What it changes
Poor Metabolizer (PM)	Can't convert prodrugs. Codeine/tramadol give no relief but full side effects. Fentanyl still hits fully.	Prodrug opioids are a trap. Extreme fentanyl-supply risk. Carry naloxone.
Intermediate (IM)	Reduced enzyme function.	Often needs dose adjustment or non-prodrug alternatives.
Normal (NM)	Standard processing.	Standard dosing assumptions apply.
Ultrarapid (UM)	Converts prodrugs dangerously fast; burns through standard antidepressants.	Codeine can be fatal. Standard antidepressant doses may be ineffective.

System 4 — GABRA2 / GABRB3 (the braking system)

This is the impulse-control axis, and it's the one most often missed. GABRA2 sets your inhibitory tone — how forcefully the 'stop' signal reaches the amygdala once a craving fires. The brake pedal exists; the linkage to the wheels is weaker than standard. This is receptor architecture, not weakness — some of the most analytically rigorous people carry it.

Result	Meaning	What it changes
GABRA2 high-risk (rs279858 T-haplotype) — ~15–20%	Structurally weak inhibition. Can see clearly that acting is a bad idea and still not halt it.	Alcohol/benzos read as 'first reliable calm.' Add the inhibitory-restoration stack. Polysubstance pattern common.
GABRA2 partial-risk — ~30–35%	Intermediate GABA tone.	Restoration stack worth implementing; watch impulse load under stress.
GABRB3 risk variant	Lower seizure threshold; governs childhood-onset risk and withdrawal severity.	Withdrawal from alcohol/benzos is more dangerous — the specific genetic basis for why it can be fatal in some and merely uncomfortable in others. Medical supervision is mandatory, not optional.

Result	Meaning	What it changes

Why the braking system matters even for stimulant users

Weak brakes are substance-agnostic. The variant was found through alcohol research, but compromised inhibition means the brain can't interrupt the craving circuit for any drug. It's the biological reason GABRA2 profiles so often present with polysubstance histories rather than a single drug — and it compounds every other system below.

The trauma multiplier: GABRA2 risk interacts with adverse childhood experiences to produce the most severe profiles — not genetics alone, not environment alone, but the collision. The good news the library insists on: the brakes can be rebuilt (magnesium, theanine, taurine, glycine, and precision-movement training), imperfectly but measurably, over months.

System 5 — OPRM1 and DRD2 (the receptor layer)

- **OPRM1 A118G.** G-allele carriers (AG/GG) need higher opioid doses for the same effect and respond better to naltrexone — directly useful when planning medication-assisted recovery.
- **DRD2 Taq1A (Reward Deficiency).** The A1 allele means ~30% fewer D2 receptors — reward deficiency at the receiving end, regardless of how much dopamine you make or clear. The library's rule: this profile 'will always need more dopamine sources than average,' and single-focus recovery tends to fail — you need multiple parallel reward sources, never one all-consuming pursuit that can collapse. Support the receptor with the brain stack (UMP + DHA + CDP-choline) to rebuild density.

Step 3 — Resolve your combined profile

The variants don't add — they multiply. These are the recognized high-risk combinations, which is where most treatment-resistant cases actually live. This is also why a single-gene read is never enough.

Combined profile	Architecture	What it looks like & the primary levers
Depleted Worrier — MTHFR T/T + COMT Met/Met	Low production + slow clearance	Depression and anxiety amplifying each other. First opioids/benzos/alcohol felt like relief, not euphoria. Levers: methylfolate 15mg, magnesium 800mg, SAMe 800mg, theanine, calm environment.
Understimulated Warrior — MTHFR T/T + COMT Val/Val	Low production + fast clearance	Anhedonia, restlessness, low motivation. Stimulants let them 'feel like the person they were supposed to be.' Levers: methylfolate 7.5mg, tyrosine, mucuna, quercetin, high-intensity exercise, Wellbutrin bridge.
Metabolic Mismatch — CYP2D6 PM/UM + MTHFR/COMT	Bad prescription match + real need	The pipeline into illicit opioids after a prescription 'failed' pharmacogenomically. Extreme fentanyl risk (fentanyl bypasses CYP2D6). Levers: fix the medication match; carry naloxone.
Impulsive Stimulant — GABRA2 + COMT Val/Val	Weak brakes + stimulation-seeking	Fast clearance drives seeking; GABA deficit prevents interrupting the craving. Levers: Warrior dopamine build + full inhibitory-restoration stack + precision-movement training.
Anxious Depressant — GABRA2 + COMT Met/Met	Weak brakes + compromised self-soothing	Most rapid progression to dependence. Levers: Sage calm stack + inhibitory restoration; medical supervision for any alcohol/benzo cessation.
Depleted-Impulsive — GABRA2 + MTHFR T/T	Weak brakes + inadequate supply	Both ends need simultaneous support: build supply and rebuild inhibition at once. Multisubstance pattern typical.

The read, in one line

Your combined profile tells you which lever is primary: MTHFR-heavy → build supply first. COMT Val/Val → feed and slightly slow dopamine. COMT Met/Met → provide calm and clearance support. GABRA2 → rebuild the brakes. CYP2D6 non-normal → fix the prescription match and carry naloxone. DRD2 A1 → support the receptor and diversify rewards. Everything in Steps 4–5 is these levers applied to a specific drug.

Step 4 — Layer in the substance

Now the part a single-substance pamphlet can't do: the same person recovers differently depending on what they got hooked on, because different drugs hijack different parts of the same system. Three worked examples across the three major mechanism classes — watch how the Step 2–3 profile changes the answer each time.

Worked example A — Cocaine (a stimulant)

Mechanism: floods dopamine 3–4× baseline. Traps Warriors and Understimulated Warriors most predictably — the stimulant was compensating for fast clearance and/or low production. Withdrawal is neurochemical crash, not medical danger, but the anhedonia is the relapse engine.

- **Profile that predicts it:** COMT Val/Val, often + MTHFR T/T, sometimes + DRD2 A1; add GABRA2 for the impulsive-binge pattern.
- **Daily direction:** Rebuild dopamine on the production side — tyrosine and mucuna for precursors, methylfolate underneath so the raw materials exist, quercetin to extend what's made. High-intensity exercise is pharmacology here, not a lifestyle tip.
- **Taper/bridge:** no medical detox required; Wellbutrin (with a prescriber) can bridge dopamine; tyrosine up during the crash window.
- **Life design:** Warrior green-light — stakes, competition, measurable progress. A gentle-yoga-only plan fails this person by understimulation.

Worked example B — Ketamine (a dissociative)

Mechanism: NMDA antagonist producing dissociation/ego-dissolution. Doesn't map cleanly onto the stimulant-vs-depressant axis, so profile predicts less about who gets trapped and more about comorbid load and the physical harm ceiling.

- **Distinct risk:** ketamine cystitis — real, dose-dependent bladder scarring that at high chronic doses can require surgery. A hard medical stop the neurochemical framework doesn't override: urological symptoms mean escalate to a doctor, not adjust a supplement.
- **Daily direction:** Foundation methylation + COMT support per the underlying profile; NAC for glutamatergic/oxidative support and craving reduction. No clean precursor-replacement move like the stimulant case, because the action is dissociative, not a neurotransmitter flood.
- **The integration point:** clinically administered ketamine with integration support is a genuinely different intervention from recreational use — the library treats the container, not just the molecule, as the variable.

Worked example C — Kratom (an opioid-type agonist)

Mechanism: 7-hydroxymitragynine is a partial mu-opioid agonist. Withdrawal is opioid-type — real, physical, 12–24h onset. Often traps people leaning Sage / Depleted Worrier who arrived via a legitimate opioid or pain problem the system didn't solve.

- **Profile that predicts it:** COMT Met/Met, often + MTHFR T/T; OPRM1 and CYP2D6 status change the medication plan.
- **Taper/bridge — the key move:** buprenorphine manages opioid-type withdrawal, kratom included, and is requestable by name at an ER or from a MAT prescriber. If

OPRM1 is G-allele, naltrexone response is better and worth raising for maintenance. Add DLPA and slow 5–10% reductions.

- **Daily direction:** Sage foundation — methylfolate, magnesium, SAMe, theanine to provide the calm the drug was providing; DLPA for endorphin support during taper. Kratom's CYP interactions can raise its effective dose — review current meds before adding anything.

Why this can't collapse into 'just fix the one substance'

The substance tells you the mechanism to counter; the DNA tells you the foundation to build. Two people quitting kratom get different stacks if one is a Warrior and one is a Depleted Worrier. One person quitting cocaine and later drinking gets the same underlying foundation with a different taper each time. Profile-first, substance-second — which is exactly why a per-drug pamphlet alone leaves the durable work undone.

Step 5 — Build the stack, taper, and life design

The output: the three-phase protocol the framework drives toward. Phase One builds biology, Phase Two tapers with that biology in place, Phase Three redesigns the life around the genotype.

Phase One — Baseline restoration (months 1–3)

The foundation everyone builds, tuned by profile. Titrate methylfolate up over ~8 weeks — too fast causes overmethylation symptoms (anxiety, insomnia).

Universal base (all profiles)

- L-methylfolate: start 1mg → 7.5–15mg over 8 weeks (dose by MTHFR severity)
- Methylcobalamin (active B12) 5,000mcg sublingual · Magnesium glycinate 400–800mg · P-5-P (B6) 50mg · TMG 1,000mg
- Omega-3 (EPA/DHA) 2–3g · NAC 1,200mg (also reduces cravings) · curcumin 1,000mg with piperine

Warrior add-on (COMT Val/Val)

- L-tyrosine 1,000–2,000mg · mucuna pruriens 200–400mg · quercetin 500mg 2×/day · green tea extract 400mg

Sage/Worrier add-on (COMT Met/Met)

- L-theanine 400mg 2×/day · SAMe 800mg · taurine 1,000mg · avoid stimulating supplements

Braking-system add-on (GABRA2/GABRB3 risk)

- Magnesium glycinate 600–800mg divided (receptor sensitivity) · L-theanine 400mg 2×/day · taurine 1,000mg · glycine 3g before bed · plus precision-movement training (below). Clinician-only bridges: gabapentin/pregabalin, baclofen, KSM-66 ashwagandha.

Phase Two — Substance taper with biological support (months 3–6)

Begin only once Phase One is established and showing measurable effect. Match the taper to the substance:

- **Opioids/kratom:** buprenorphine with a MAT prescriber; slow 5–10% reductions every 1–2 weeks; DLPA 500mg 2×/day; black seed oil for tolerance.
- **Stimulants/cocaine:** Wellbutrin bridge (with prescriber); tyrosine up to 2,000mg 2×/day; high-intensity exercise begins now.
- **Alcohol/benzos (and any GABRB3-risk profile):** medical supervision is mandatory — withdrawal can seize and kill. Detox first, then apply the protocol. GABA support (theanine, taurine, magnesium, glycine); gabapentin/baclofen as prescriber-guided bridges.

Phase Three — Environmental & behavioral redesign (months 6–12)

Genotype-specific life design, because matched activity changes gene expression (BDNF, D2 density, cortisol sensitivity) over months — the part that makes the substance irrelevant rather than merely resisted.

	Warrior (Val/Val)	Sage/Worrier (Met/Met)	GABRA2 (weak brakes)
Exercise	HIIT, powerlifting, martial arts, competitive sport	Yoga, zone-2 cardio, swimming, climbing	Precision movement (Pilates/yoga as controlled inhibition), zone-2 cardio, 4-7-8 breathing
Avoid	Slow solo cardio → rumination	HIIT/chaotic competition → cortisol flood	Uncontrolled high-arousal chaos without the precision element
Social	A squad to compete with	A small guild built on shared creation	Structure that rehearses the pause; martial arts if also Warrior

The honest timeline — for a substance-damaged brain (the 'addict virgin' correction)

Standard recovery timelines were measured on lightly-addicted or non-addicted people. Your starting point is different, and the guide is explicit about it: 'exercise improves mood in 2 weeks' becomes 4–8 weeks; '8-week meditation benefit' becomes 12–16 weeks at a starting dose of 2–5 minutes built up slowly. The improvement may be happening physiologically before you feel it.

Why: the reward and prefrontal machinery those studies assumed intact is exactly what severe addiction impaired. Exercise-reduces-craving needs a prefrontal cortex able to act on the reduced signal — so early on it often needs pairing with NAC, the methylation stack, and (a tier the standard stack omits) nootropic support for AMPA/BDNF repair. The interventions work. They work more slowly for you. That is not failure — it's the correct timeline for your actual starting point.

Step 6 — Drive the whole thing with AI

The framework is designed to run through an AI assistant as a personalized reasoning partner — that's what turns this static guide into something that responds to your actual results. Build the profile once, paste it at the start of any recovery-related AI conversation. Without it, answers are generic; with it, they account for your variants, history, medications, and triggers.

=== MY RECOVERY KERNEL PROFILE ===

GENETIC VARIANTS: MTHFR C677T ___ · MTHFR A1298C ___ · COMT Val158Met ___ (Warrior/Balanced/Sage) · CYP2D6 ___ (PM/IM/NM/UM) · CYP2C19 ___ · GABRA2 ___ · GABRB3 ___ · OPRM1 ___ · DRD2 ___

LAB VALUES: Homocysteine ___ (goal <10) · B12 ___ (goal >400) · Vitamin D ___ (goal 40–60)

SUBSTANCE HISTORY: primary substance(s) ___ · duration ___ · current status ___ · prior treatment ___

CURRENT MEDICATIONS: ___ CURRENT SUPPLEMENTS (dose + timing): ___

KNOWN TRIGGERS: ___

=== END PROFILE ===

The four prompts that do the work

- **Genetic interpretation:** 'Given these variants, per CPIC/FDA pharmacogenomic guidance — predicted response to [medication], which supplements to prioritize, which substances to avoid for my CYP status, and the most-supported lifestyle changes?'
- **Medication safety review:** 'I've been prescribed [drug] at [dose]; CYP2D6 is [status]; here are my meds/supplements — appropriate for my metabolizer status? dose change? interactions? alternatives if contraindicated?'
- **Supplement stack optimization:** 'Here's my profile, current stack, symptom ratings, monthly budget — what to add, what's unnecessary, optimal timing, interactions, and the top three if budget's tight?'
- **Weekly check-in:** 'This week's 1–10 ratings for mood/energy/sleep/craving/stress plus adherence — patterns, adjustments, warning signs, next week's priority?' Direction across weeks matters more than any single week.

The boundary that doesn't move

AI is an educational and reasoning tool, not a treatment provider. It can interpret variants, check interactions, and help you write precise questions for a prescriber. It cannot prescribe, cannot handle emergencies, and cannot override clinical judgment.

Severe craving → support person or crisis line first. Suicidal thoughts → 988. Medical emergency → 911. AI is for informed, stable decision-making — not crisis.

The whole guide in one line: sequence your genome (or estimate it free), read your six gene systems, resolve your combined profile, layer in your substance, build the Phase-One stack before you taper, expect the honest slower timeline, and run it all through an AI profile you keep current. The DNA sets the foundation; the substance sets the counter-move; the two together are the protocol.